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All Sealed Endoleaks are not the Same: A Treatment Strategy Based on an *ex-vivo* Analysis†

M. Mehta, T. Ohki*, F. J. Veith and E. C. Lipsitz

Division of Vascular Surgery, Montefiore Medical Center and Albert Einstein College of Medicine

Purpose: *factors contributing to pressure transmission through thrombosed or sealed endoleaks have not been elucidated. The purpose of this investigation was to create an* ex-vivo *model that mimics patent and sealed endoleaks and that can quantitatively analyse the effects of length, diameter and thrombus on pressure transmission to the interior of the aneurysm sac.*
Methods: *in the* ex-vivo *model, endoleak channels (ELCs) of various lengths (2 cm, 6 cm, 10 cm) and diameters (0.6 cm, 1.0 cm, 1.4 cm) were constructed using polytetrafluoroethylene (PTFE) grafts and attached to an artificial aneurysm sac. These ELCs were incorporated within a mock circulation made of rubber tubing connected to a pulsatile pump. Peak systolic pressure (PSP) was recorded in the aneurysm sac, distal to each ELC. Subsequently the ELCs were filled with human thrombus, and the pressure measurements repeated* ($n=5$). *Data was evaluated by regression analysis.*
Results: *peak systolic pressure in the artificial circulation was maintained at 150 mmHg. In the absence of thrombus pressure did not change across the ELC, regardless of its length or diameter. In the presence of organised thrombus, the pressure curves distal to the ELC were dampened, and the pressure reduction was directly proportional to the length and inversely proportional to the diameter of the ELC. Regression analysis indicated statistical significance.*
Conclusions: *in the absence of thrombosis, pressure transmitted via an ELC to the aneurysm sac is unchanged regardless of its length or diameter. All sealed endoleaks also transmit pressure. However, when an endoleak has thrombosed, pressure reduction is directly proportional to the length and inversely proportional to the diameter of its channel. This* ex-vivo *model suggests that Type 2 endoleaks with longer channels and smaller diameters would derive a greater benefit from adjunctive manoeuvres (coil embolisation) that hasten thrombosis. On the other hand, thrombosis of endoleaks with short and wide channels (e.g. Type 1) may not result in substantial pressure reduction within the aneurysm sac and a successful outcome.*

Key Words: Sealed endoleaks.

Introduction

Although endovascular aortic aneurysm repair (EVAR) has gained wide acceptance, endoleaks continue to be among the most common complications of the procedure. Endoleaks, defined as perigraft blood flow within the aneurysm sac, are associated with incomplete sealing of proximal and distal fixation sites, retrograde flow from collateral branches, defects in the stent-graft itself, or graft porosity.[1–3] We are beginning to understand the natural history and pathophysiologic significance of patent endoleaks. However, the significance of so-called "sealed" endoleaks remains poorly understood and there is no consensus regarding its treatment options, with one exception; sealed endoleaks that lead to aneurysm enlargement necessitate treatment.

Options for treating endoleaks include coil embolisation, addition of stent-graft cuffs and extensions, endoscopic ligation of inferior mesenteric and lumbar arteries, redo endovascular stent-graft repair, and open surgical repair. Factors contributing to pressure transmission through thrombosed endoleaks, which will affect the treatment strategy for patent endoleaks, have not been elucidated. The purpose of this study was to analyse the effects of length, and diameter of the ELC on pressure transmission to the aneurysm sac when the channel was patent and when it was thrombosed or "sealed".

Materials and Methods

The experiments were performed in an *ex-vivo* model, which consisted of an artificial circulation made of

* Please address all correspondence to: T. Ohki, Division of Vascular Surgery, Montefiore Medical Center, 111 East 210th St., New York, NY 10467, U.S.A.
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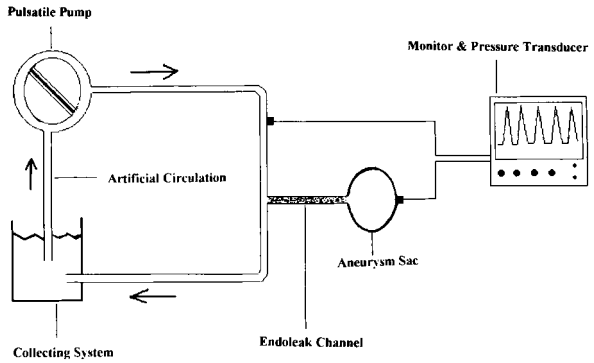

Fig. 1. *Ex-vivo* model consisting of an artificial systemic circulation made of rubber tubing connected to a pulsatile pump (Haemonetics; Braintree, MA, U.S.A.) and a collecting system. Endoleak channels of various lengths (2 cm, 6 cm, 10 cm) and diameters (0.6 cm, 1.0 cm, 1.4 cm), constructed using polytetrafluoroethylene grafts (PTFE; W.L. Gore & Associates, Flagstaff, AR, U.S.A.), were attached to an artificial aneurysm sac without outflow and incorporated within the mock circulation.

rubber tubing (diameter: 15 mm, length: 150 cm) connected to a pulsatile pump (Haemonetics; Braintree, MA, U.S.A.) and a collecting system. Endoleak channels (ELCs) of various lengths (2 cm, 6 cm, 10 cm) and diameters (6 mm, 10 mm, 14 mm), constructed using polytetrafluoroethylene grafts (PTFE; W.L. Gore & Associates, Flagstaff, AR, U.S.A.), were incorporated within the artificial circulation (Figs 1, 2). An artificial aneurysm sac made of silicone tubing was attached to the end of each ELC. Heparinised canine blood was used within the artificial circuit. The rubber tubing created compliance within the system, and resistance was adjusted by controlling the outflow diameter. Frequency of the pulsatile pump was adjusted to maintain a pulse rate of 80 beats/min, and flow at 5 l/min.

A pressure transducer (Konigsberg Inst. Inc., Pasadena, CA, U.S.A.) that permits frequency response and sensitivity, in conjunction with an analog-digital board (Metrabyte DAS-1407; Trinton Technologies, San Diego, CA, U.S.A.), was implanted in the wall of the aneurysm sac, without outflow. Peak systolic pressures were recorded proximal and distal to each ELC ($n=5$). Subsequently, the ELCs were filled with organised human thrombus, obtained on the day of experiments during open abdominal aortic aneurysm repair, and the pressure measurements repeated ($n=5$).

Statistical analysis was performed by regression analysis. The data were expressed as mean value $\pm$ standard deviation. Statistical significance was assumed at the 95% confidence interval (R^2 >0.95).

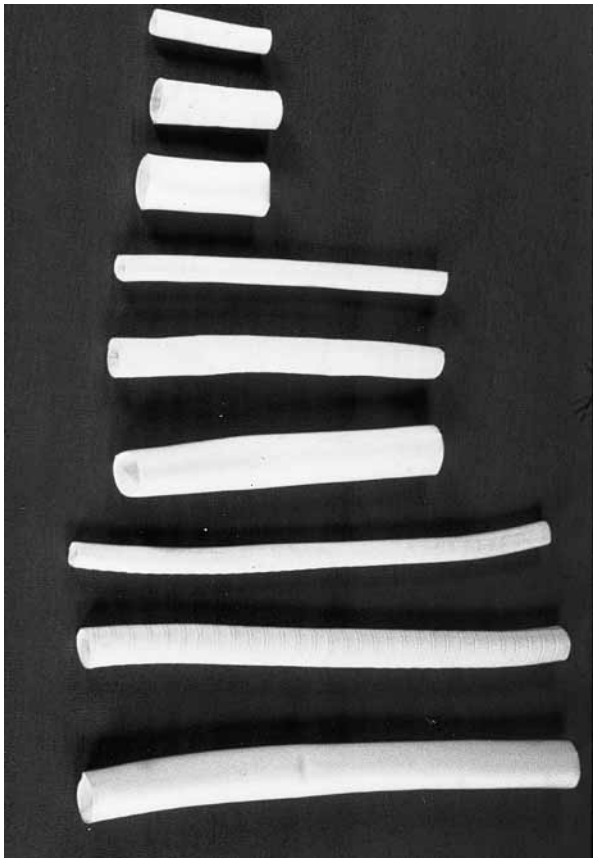

Fig. 2. Endoleak channels of various lengths (2 cm, 6 cm, 10 cm) and diameters (0.6 cm, 1.0 cm, 1.4 cm).

Results

Peak systolic pressure in the artificial circulation was maintained at 150 mmHg. In the absence of thrombus, pressure did not change across the ELCs, regardless of their length or diameter. In the presence of organised thrombus the pressure curves distal to the ELCs, in the aneurysm sac, were dampened and the pressure reduction was directly proportional to the length and inversely proportional to the diameter of each ELC (Fig. 3). A detailed listing of pressure measurements in the systemic circulation and in the aneurysm sac, proximal and distal to the ELCs, with and without the presence of thrombus, is recorded in Table 1.

Discussion

The goal of endovascular repair is to successfully exclude the aneurysm from systemic pressure and to prevent rupture. Patent endoleaks can transmit systemic pressure to the aneurysm sac and their incidence can range from 12–44%.[4–6] The clinical sig-

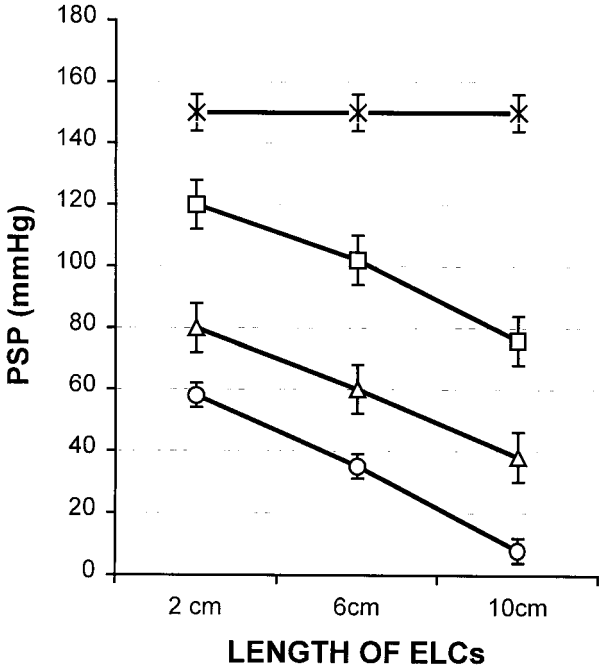

Fig. 3. Pressure transmission across patent and thrombosed endoleak channels depending on their length and diameter. (*) Patent ELCs, diameter: 1.4, 1.0, 0.6 cm. (□) Sealed ELCs, diameter: 1.4 cm. (△) Sealed ELCs, diameter: 1.0 cm. (○) Sealed ELCs, diameter: 0.6 cm.

Table 1. Peak systolic pressures (PSP) proximal to the endoleak channel (ELC) within the circulation were maintained at 150 ± 5 mmHg. PSP distal to the ELCs within the aneurysm sac are listed below.

ELC Diameter	ELC Length	PSP without Thrombus (mmHg)	PSP with Thrombus (mmHg)
1.4 cm	2 cm	150 ± 5	120 ± 4
1.4 cm	6 cm	150 ± 5	102 ± 4
1.4 cm	10 cm	150 ± 5	76 ± 4
1.0 cm	2 cm	150 ± 5	80 ± 4
1.0 cm	6 cm	150 ± 5	60 ± 3
1.0 cm	10 cm	150 ± 5	38 ± 2
0.6 cm	2 cm	150 ± 5	58 ± 3
0.6 cm	6 cm	150 ± 5	35 ± 2
0.6 cm	10 cm	150 ± 5	8 ± 2

nificance of endoleaks remains poorly understood and their unpredictable nature in the endovascular treatment of aortic aneurysms is a topic of controversy. Several investigators have attempted adjunctive manoeuvres to induce thrombosis of the ELC, hoping to eliminate pressurisation of the aneurysm sac. However, others have suggested that even thrombosed or sealed endoleaks may result in persistent pressurisation of the aneurysm sac (endotension), and can be associated with an increased risk of aneurysm rupture.[7,8] Recently clinical as well as bench-top studies have shown that patent endoleaks, regardless of their size, transmit systemic pressures to the aneurysm sac.[9,10] However, factors contributing to pressure transmission through sealed endoleaks have not been elucidated.

The results of our experiments indicate that all endoleaks, even when sealed, transmit some pressure. In the absence of thrombosis, pressure transmitted through an ELC to the aneurysm sac without outflow is systemic and unchanged regardless of its length or diameter. However, when an endoleak has sealed, pressure reduction is substantially influenced by the length and diameter of its channel. Sealed endoleaks with longer channels and smaller diameters (e.g. Type II endoleaks) have a greater pressure reduction across them when compared to endoleaks with shorter channels and larger diameters (e.g. Type I or Type III endoleaks).

Identifying some of the mechanisms involved in pressure transmission through endoleaks helps in guiding appropriate treatment options for various endoleaks. For instance, several investigators have adopted algorithms to treat all endoleaks by coil embolisation to achieve thrombosis. Kato *et al.* achieved radiographic seals after coil embolising 10 Type I, II, and III endoleaks that persisted after endovascular aneurysm repair.[11] Makaroun *et al.* reported their experience with coil embolising all persistent endoleaks at 6 months after endovascular abdominal aortic aneurysm repair. Six of the patients underwent coil embolisation of Type I endoleaks, all achieving a radiographic seal.[12] However, these investigators did not measure pressures in the excluded aneurysm sacs and failed to demonstrate freedom from endotension. Gorich *et al.* reported a similar experience with coil embolisation of 10 Type I and 11 Type II endoleaks.[13] They too achieved radiographic seals, yet failed to comment on pressure reduction within the aneurysm sac.

The concept of endotension is important because coil embolisation of endoleaks may result in obliteration of endoleaks but may not necessarily reduce aneurysm sac pressurisation, particularly when the ELCs have short lengths and large diameters. Thrombus may seal the aneurysm sac or the ELC resulting in an inability of computerised tomography, contrast arteriography, or ultrasonography to detect the endoleak. However, the aneurysm might be subjected to continuous high pressure, increasing its risk of rupture. In the past we have investigated the relationship between endoleaks, intra-aneurysmal pressure, and the effects of coil embolisation.[14] In those experiments, regardless of achiev-

ing a complete seal of stent-graft related endoleaks with a short length and a large diameter (Type III), aneurysm sac pressures did not decrease substantially. Thus our previous results are consistent with the findings in our present experiments.

Our present results suggest that pressure transmission through patent ELCs to the aneurysm sac is unchanged regardless of its length and diameter. Furthermore, when an endoleak has sealed, endotension is directly proportional to the length and inversely proportional to the diameter of its channel. Some of these findings are supported by recent papers by Baum *et al.*, who evaluated pressure transmission through endoleaks and the role of coil embolisation of lumbar and inferior mesenteric artery endoleaks (Type II).[9,15] Their results suggest that all patent endoleaks transmit systemic pressure to the aneurysm sac, and coil embolisation of Type II endoleaks reduces pressure transmission significantly.

Freedom from endoleaks remains an unreliable guide to treatment success. Our results in the *ex-vivo* model suggests that patients with Type II endoleaks with longer channels and smaller diameters would benefit more from adjunctive manoeuvres, such as coil embolisation that hasten thrombosis. On the other hand, thrombosis of endoleaks with short and wide channels (e.g. Type I or Type III) may not result in a substantial pressure reduction and a successful outcome. In such cases we recommend placement of a proximal cuff, a supplementary graft limb, or open repair to obliterate the leak and lower the pressure within the aneurysm.

Acknowledgements

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